


Molecular mimicry of SARS-nCoV-2 spike to human proteins including thrombopoietin and TLR-8

High autoimmune potential shown via cross-reacting antibodies.



Jessica Rose

Jul 4

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Please refer to the paper entitled: “[Potential Autoimmunity Resulting from Molecular Mimicry between SARS-CoV-2 Spike and Human Proteins](#)” published June 28, 2022 in [Viruses](#).¹

This was brought to my attention via an [article](#) I posted yesterday. They found the following.

We discovered molecular mimicry hotspots in Spike and highlight two examples with tentative high autoimmune potential and implications for understanding COVID-19 complications. We show that a TQLPP motif in Spike and thrombopoietin shares similar antibody binding properties. Antibodies cross-reacting with thrombopoietin may induce [thrombocytopenia](#), a condition observed in COVID-19 patients.

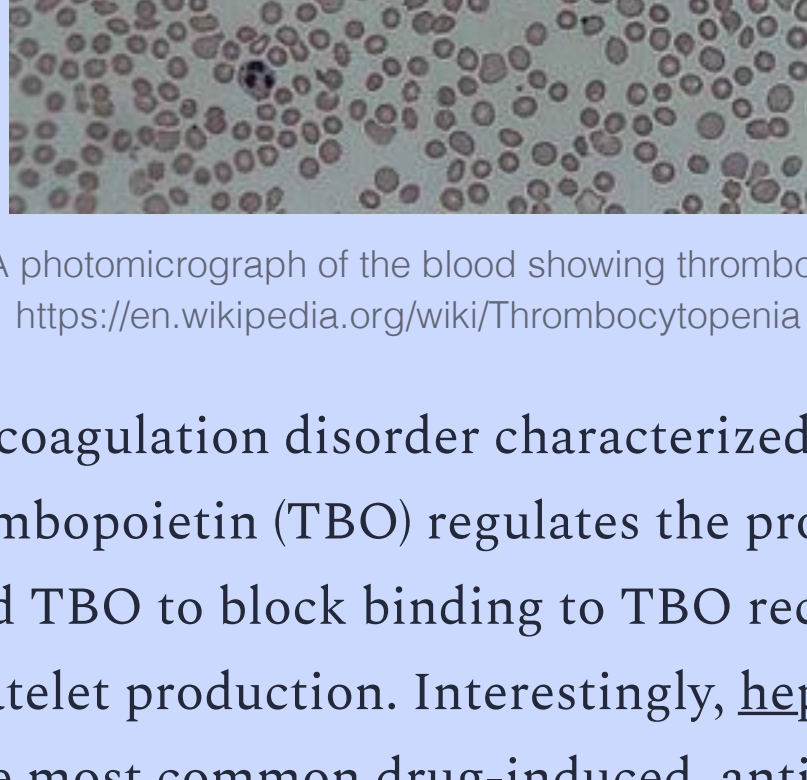


Figure 1: A photomicrograph of the blood showing thrombocytopenia. <https://en.wikipedia.org/wiki/Thrombocytopenia>

Thrombocytopenia is a coagulation disorder characterized by abnormally low levels of platelets. Thrombopoietin (TBO) regulates the production of platelets.^{2,3} The autoantibodies bind TBO to block binding to TBO receptors and subsequently inhibit platelet production. Interestingly, [heparin-induced thrombocytopenia](#) is the most common drug-induced, antibody-mediated thrombocytopenic disorder.⁴

The authors summarize a list of motifs that share this potential for induction of autoimmunity from other proteins. There are a total of 16 human proteins identified as 3D mimics based on an RMSD ([Root Mean Square Deviation](#) - small differences between values) of at most 1 Å (Ångstrom) listed in Table 1. Of these is thrombopoietin - a regulator of platelet production, and Toll-Like Receptor 8 (TLR-8). TLR-8 is a receptor that recognizes single stranded RNA (ssRNA) and when it does, it recruits [MyD88](#) and leads to activation of the transcription factor [NF-κB](#) (DNA transcription) to initiate an antiviral response. TLR-8 recognizes ssRNA viruses such as HIV and HCV, and since coronaviruses are also ssRNA viruses, it likely recognizes them too.⁵ It is also interesting to note that TLR-8 expression levels are also significantly elevated in individuals with severe and critical COVID-19.⁶ This implies that cross-reactivity, if possible, would be more likely to ensue.

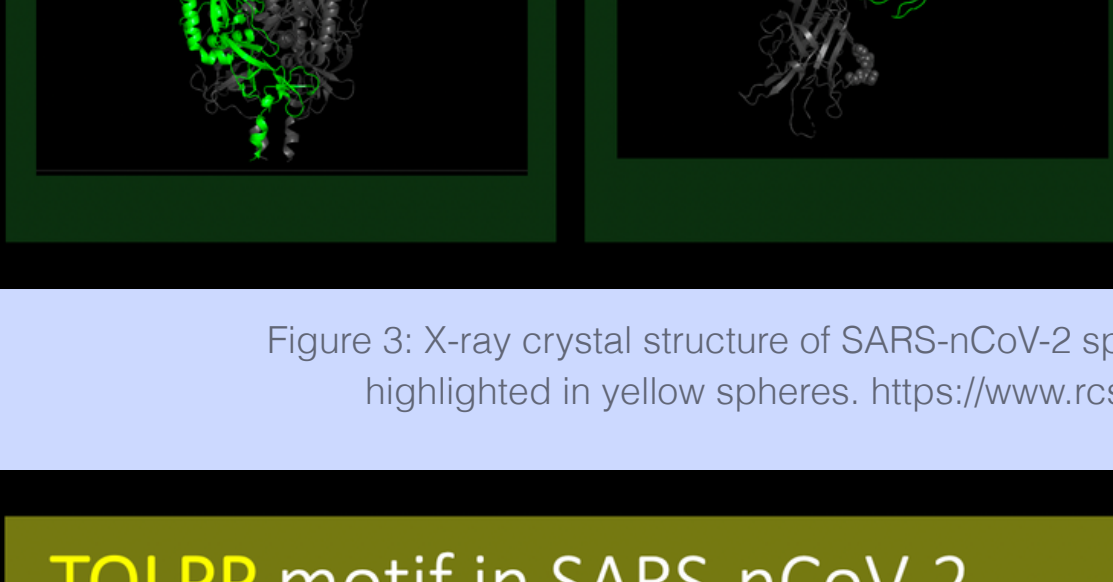
Table 1. 3D-mimics found for SARS-CoV-2 Spike.						
Motif	Protein	Species	RMSD (Å)	Z-Score	EpiScore	PDB_Chain
TQLPP	Thrombopoietin	Human	0.46	-1.34	10.87	1V7N_X
QLPPA	SMYD3 protein	Human	0.38	-1.42	13.16	5CCL_A
KNLRE	Toll-like receptor 8	Human	0.87	-0.92	5.75	6WML_D
FTVEKG	Pollen allergen Phl p2	<i>Phleum pratense</i>	0.76	-1.03	7.89	1WHP_A
GEVFN	Integrin beta 1	Human	0.63	-1.16	7.94	7NWL_B
HAPAT	Activator of 90 kDa heat shock protein	Human	0.74	-1.05	6.76	7DME_A
YSTGS	ATPase homolog 1	Human	0.48	-1.31	10.42	1K62_B
EHVNN	Argininosuccinate lyase	Human	0.29	-1.21	17.24	2ZJW_A
NLLIQ	Cas6 protein 2 alpha isoform	Human	0.57	-1.22	8.77	5C51_A
LLOYG	DNA polymerase subunit gamma 1	Human	0.20	-1.60	25.00	1N11_A
LPDPS	BRCA1-A complex subunit BRE	Human	0.32	-1.48	15.62	6CVW_C
LDPS	Semaphorin 7a	Human	0.84	-0.91	5.95	3NVQ_A
DPSKP	60S ribosomal protein L3	Human	0.10	-1.70	50.00	4LUS_B
DPSKP	Alanine and proline-rich secreted protein apa precursor	<i>Mycobacterium tuberculosis</i>	0.21	-1.59	23.81	5ZXA_A
IAARD	Talin	<i>Mus musculus</i>	0.74	-1.05	6.76	6R9T_A
GNCDV	Tryptophan-tRNA ligase	Human	0.91	-0.88	5.49	1OST_A
SFKEE	Small subunit processome component	Human	0.32	-1.48	15.62	7MQA_SP
EELDK	20 homolog	Human	0.22	-1.58	22.73	7MZF_A
ELDKY	Kynureninase	Human	0.12	-1.68	41.67	6EAE_F
DKYFK	Fusion glycoprotein F0	Respiratory syncytial virus	0.14	-1.66	35.71	4N78_A
DKYFK	Cytoplasmic FMRI-interacting protein 1	Human	0.14	-1.66	35.71	4N78_A

Figure 2: 3D-mimics found for SARS-nCoV-2 spike. Table 1 from paper.

These are alarming findings, but I think I can add to the alarm, if I am correct that is. One of the motifs implicated in autoimmune reactions via molecular mimicry found in SARS-nCoV-2 and human thrombopoietin, namely: TQLPP, is **not** found in SARS-nCoV. Now, where have I heard this kind of story before? Furin cleavage site? Other inserts? Hmm. One of the clinical manifestations of SARS-nCoV-2 that appears distinct from SARS-nCoV is this bleeding/clotting/vascular component. What if the antibodies generated against this motif in the spike protein of SARS-nCoV-2 are going after human thrombopoietin and via cross-reactivity inducing down-stream signaling to modify platelet production?

The implications of this are quite staggering because it is highly unlikely (some genius out there can calculate the likelihood - ahem: Jikky (his fan account got erased from Twitter today again)) that this motif would ‘arise’ in the SARS-nCoV-2 version ‘naturally’. It’s another brick in the wall. In my opinion, it’s statistically impossible for all of these unique SARS-nCoV-2 motifs to naturally arise.

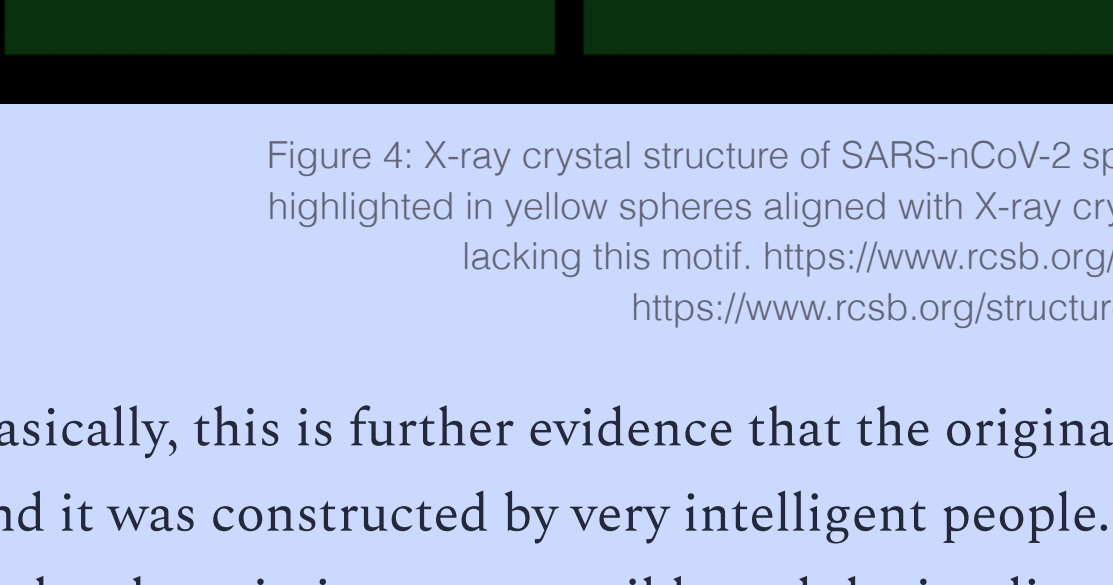
TQLPP motif in Spike shown in yellow spheres – 3 perspectives



- The site is exposed and makes it a candidate for binding
- Is this motif in SARS-nCoV? No.

Figure 3: X-ray crystal structure of SARS-nCoV-2 spike protein with TQLPP motif highlighted in yellow spheres. <https://www.rcsb.org/structure/7CWM>

TQLPP motif in SARS-nCoV-2 (green) *not* found in SARS-nCoV (pink)



- It is highly unlikely that this motif implicated in autoimmunity via molecular mimicry would arise naturally – like the furin cleavage site.

Figure 4: X-ray crystal structure of SARS-nCoV-2 spike protein with TQLPP motif highlighted in yellow spheres aligned with X-ray crystal structure of SARS-nCoV lacking this motif. <https://www.rcsb.org/structure/7CWM>; <https://www.rcsb.org/structure/5XS8>

Basically, this is further evidence that the original SARS-nCoV-2 was constructed, and it was constructed by very intelligent people. They must have known that this molecular mimicry was possible and the implications of autoimmune responses. We are seeing ITP, TTP and all sorts of thrombocytopenias in VAERS and in life.

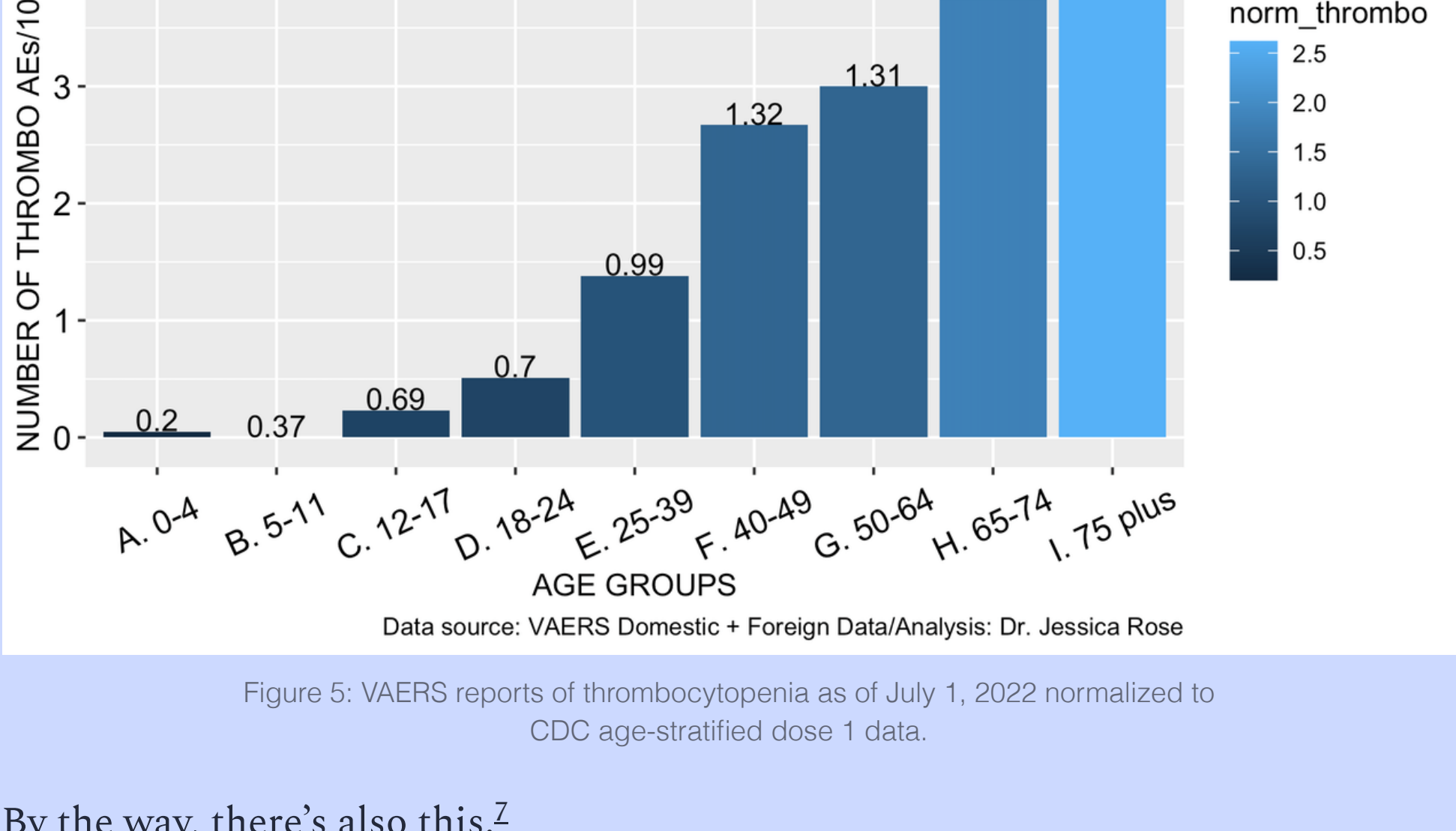


Figure 5: VAERS reports of thrombocytopenia as of July 1, 2022 normalized to CDC age-stratified dose 1 data.

By the way, there’s also [this](#).⁷

Results:

SARS-CoV-2 spike glycoprotein was found to share 41 minimal immune determinants, that is, pentapeptides, with 27 human proteins that relate to oogenesis, uterine receptivity, decidualization, and placentation. All the shared pentapeptides that we identified, with the exception of four, are also present in SARS-CoV-2 spike glycoprotein-derived epitopes that have been experimentally validated as immunoreactive.

We’re really up the creek without a paddle on this one.

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YZRJul 4

Hmm. These spike proteins with anti-oogenic autoimmune mimicry just happen to be administered via the LNP that just happen have an affinity for the ovaries. Almost as if this were planned. 🤔

♡48ReplyCollapse

6 replies
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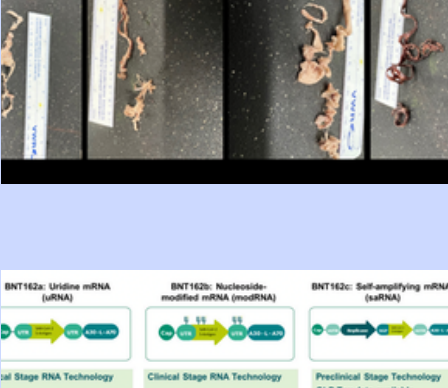
steveBDHJul 4

Thanks Dr. Rose. Have you seen/read this article? https://wmcresearch.substack.com/p/urgent-warning-the-spike-protein?utm_source=substack&utm_medium=email It is difficult, to say the least, to understand what is going on if you do not have the requisite background. Thanks for all the great posts and for all the amazing work you are doing.

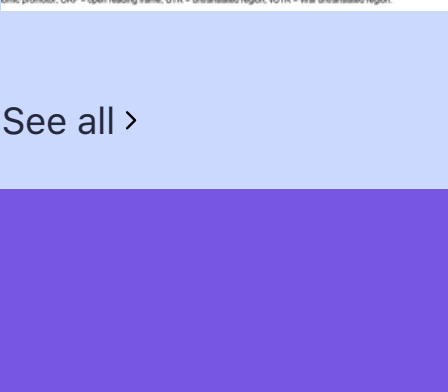
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A Report on Myocarditis Adverse Events (VAERS) in the Association with COVID-19 Injectable...
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Rewrite: Let's tag team this until everybody understands
The modified spike protein is dangerous and for very specific reasons.
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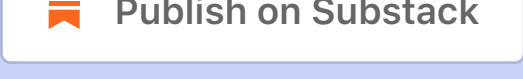
When you hear BNT162c(2), run, don't walk, RUN away.
It's already in the clinical 'trials'
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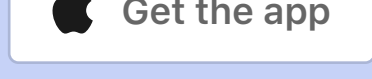
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